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L1	RGS4	26	L1

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L3 ANSWER 1 OF 12 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 2003299456 EMBASE
TI Erratum: Association and linkage analyses of ***RGS4*** polymosphisms in ***schizophrenia*** (Human Molecular Genetics (2002) vol. 11 (1373-1380)).
AU Chowdari K.V.; Mirnics K.; Semwal P.; Wood J.; Lawrence E.; Bhatia T.; Deshpande S.N.; Thelma B.K.; Ferrell R.E.; Middleton F.A.; Devlin B.; Levitt P.; Lewis D.A.; Nimgaonkar V.L.
SO Human Molecular Genetics, (15 Jul 2003) 12/14 (1781). ISSN: 0964-6906 CODEN: HMGEE5
CY United Kingdom
DT Journal; Errata
FS 022 Human Genetics
LA English

L3 ANSWER 2 OF 12 CAPLUS COPYRIGHT 2003 ACS on STN
AN 2003:518293 CAPLUS
TI Association and linkage analyses of ***RGS4*** polymorphisms in ***schizophrenia***
AU Chowdari, Kodavali V.; Mirnics, Karoly; Semwal, Prachi; Wood, Joel; Lawrence, Elizabeth; Bhatia, Triptish; Deshpande, Smita N.; Thelma, B. K.; Ferrell, Robert E.; Middleton, Frank A.; Devlin, Bernie; Levitt, Pat; Lewis, David A.; Nimgaonkar, Vishwajit L.
SO Human Molecular Genetics (2003), 12(14), 1781 CODEN: HMGEE5; ISSN: 0964-6906
PB Oxford University Press
DT Journal; Errata
LA English
AB Unavailable

L3 ANSWER 3 OF 12 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 1
AN 2003414730 EMBASE
TI Recent advances in the genetics of ***schizophrenia*** .
AU O'Donovan M.C.; Williams N.M.; Owen M.J.
CS M.J. Owen, Department of Psychological Medicine, Neuropsychiatric Genetics Unit, Univ. of Wales College of Medicine, Health Park, Cardiff CF14 4XN, United Kingdom. owenmj@cf.ac.uk
SO Human Molecular Genetics, (15 Oct 2003) 12/REV. ISS. 2 (R125-R133). Refs: 93 ISSN: 0964-6906 CODEN: HMGEE5
CY United Kingdom
DT Journal; General Review
FS 005 General Pathology and Pathological Anatomy
022 Human Genetics
032 Psychiatry
LA English
SL English
AB The high heritability of ***schizophrenia*** has stimulated much work aimed at identifying susceptibility genes using positional genetics. As a result, several strong and well-established linkages have emerged. Three of the best-supported regions are 6p24-22, 1q21-22 and 13q32-34 where single studies have achieved genome-wide significance at P<0.05 and suggestive positive findings have also been reported in other samples. Other promising regions include 8p21-22, 6q21-25, 22q11-12, 5q21-q33, 10p15-p11 and 1q42. Recently, evidence implicating individual genes within some of the linked regions has been reported and more importantly replicated. Currently, the weight of evidence supports NRG1 and DTNBP1 as ***schizophrenia*** susceptibility loci, though work remains before we understand precisely how genetic variation at each locus confers susceptibility and protection. The evidence for COMT, ***RGS4*** and G72 is promising but not yet persuasive. While it is essential that further replications are established, the respective contributions of each gene, relationships with aspects of the phenotype, the possibility of epistatic interactions between genes and functional interactions between the gene products will all need investigation. The ability of positional genetics to implicate novel genes and pathways will open up new vistas for neurobiological research, and all the signs are that genetic research is poised to deliver crucial insights into the nature of ***schizophrenia*** .

L3 ANSWER 4 OF 12 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 2
AN 2003407681 EMBASE
TI Toward ***schizophrenia*** genes: Genetics and transcriptome.
AU Ito C.; Ouchi Y.
CS C. Ito, Department of Psychiatry, Tohoku Univ. Grad. Sch. of Medicine, 1-1, Seiryo-machi, Aoba-ku, Sendai, 980-8574, Japan. cito@mail.cc.tohoku.ac.jp
SO Drug Development Research, (1 Oct 2003) 60/2 (111-118). Refs: 58 ISSN: 0272-4391 CODEN: DDREDK
CY United States
DT Journal; General Review
FS 022 Human Genetics
032 Psychiatry
LA English
SL English
AB ***Schizophrenia*** is highly heritable, and the identification of its genetic factors is receiving great attention. In this review, we digested current genetic findings about ***schizophrenia*** . At first, DISC-1 and -2 derived from a large Scottish family, and velo-cardio-facial syndrome caused by microdeletion of 22q11, are hot topics in cytogenic studies. Secondly, selections and classifications of samples become the biggest problems in linkage studies, because many following studies failed to confirm replicated linkages. Thirdly, a lot of functional and/or

positional candidate genes are analyzed. Some genes, including NRG1, DTNBP1, G72, DAAO, ***RGS4*** , and PRODH2, are recently identified in association studies. In the last method, the novel way to select functional candidate genes with a transcriptome analysis is getting presented. The transcriptome analysis makes it possible to identify up- and down-regulated genes from overall transcriptions (mRNAs). We also have an ongoing case-control study about ***schizophrenia*** , following serial analysis of gene expression (SAGE), one of the transcriptome analyses. We analyzed changes of gene expressions in methamphetamine- and phencyclidine-treated rodent cerebral cortexes, two well-known animal models of ***schizophrenia*** . We identified their homologous 209 human genes as candidates. These findings will bring us new understanding of pathophysiologic aspects and further drug targets toward ***schizophrenia*** . .COPYRGT. 2003 Wiley-Liss, Inc.

L3 ANSWER 5 OF 12 CAPLUS COPYRIGHT 2003 ACS on STN
AN 2002:158038 CAPLUS
DN 136:214957
TI Expression of the gene for regulator of G-protein signaling 4 as a diagnostic indicator for ***schizophrenia***
IN Levitt, Pat R.; Mirnics, Karoly; Kodavali, Venkata Chowdari; Nimgaonkar, Vishwajit L.
PA University of Pittsburgh, USA
SO PCT Int. Appl., 112 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002016653	A2	20020228	WO 2001-US26622	20010824
WO 2002016653	A3	20030703		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
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AU 2001088416	A5	20020304	AU 2001-88416	20010824
US 2003113721	A1	20030619	US 2001-939209	20010824
PRAI US 2000-228021P	P	20000824		
WO 2001-US26622	W	20010824		

AB A method of diagnosing, assessing susceptibility, and/or treating ***schizophrenia*** involving the observation of regulator of G-protein signaling 4 (***RGS4***) levels in a subject. Embodiments of the present invention include increasing ***RGS4*** expression levels in the cortex, either by chem. means or by genetic complementation (e.g. gene therapy). Microarray anal. of gene expression found that the transcript for the regulator of G-protein signaling 4 (***RGS4***) was consistently present at lowered levels in the prefrontal cortex of all ***schizophrenic*** subjects examd. Other members of the RGS family did not show changes in levels and transcripts of genes assocd. with G protein signaling as a whole were unaffected by the disease. Almost 30 single nucleotide polymorphisms are obsd. in the gene.

L3 ANSWER 6 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 3
AN 2002:352532 BIOSIS
DN PREV200200352532
TI Association and linkage analyses of ***RGS4*** polymorphisms in ***schizophrenia*** .
AU Chowdari, Kodavali V.; Mirnics, Karoly; Semwal, Prachi; Wood, Joel; Lawrence, Elizabeth; Bhatia, Triptish; Deshpande, Smita N.; Thelma B. K.; Ferrell, Robert E.; Middleton, Frank A.; Devlin, Bernie; Levitt, Pat; Lewis, David A.; Nimgaonkar, Vishwajit L. [Reprint author]
CS 3811 O'Hara St, Room 443, Pittsburgh, PA, 15213, USA
nimga@pitt.edu
SO Human Molecular Genetics, (1 June, 2002) Vol. 11, No. 12, pp. 1373-1380. print.
ISSN: 0964-6906.
DT Article
LA English
ED Entered STN: 26 Jun 2002
Last Updated on STN: 26 Jun 2002
AB Gene expression analyses of postmortem cerebral cortex suggest that transcription of the regulator of G-protein signaling 4 (***RGS4***) is decreased in a diagnosis-specific manner in subjects with ***schizophrenia*** . To evaluate the possible role of ***RGS4*** in the pathogenesis of ***schizophrenia*** , we conducted genetic association and linkage studies using samples ascertained independently in Pittsburgh and New Delhi and by the NIMH Collaborative Genetics Initiative. Using the transmission disequilibrium test, significant transmission distortion was observed in the Pittsburgh and NIMH samples. Among single-nucleotide polymorphisms (SNPs) spanning approximately 300 kb, significant associations involved four SNPs localized to a 10 kb region at ***RGS4*** , but the associated haplotypes differed. A trend for transmission distortion was also present in the Indian sample for haplotypes incorporating the same SNPs. Consistent with the linkage/association observed from the family-based tests, samples with affected siblings (NIMH, India) showed higher levels of allele sharing, identical by descent, at ***RGS4*** . When the US patients were contrasted to two population-based control samples, however, no significant differences were observed. To check the specificity of the transmission bias, we therefore examined US families with bipolar I disorder (BD1) probands. This sample also showed a trend for transmission distortion, and differed significantly from the population-based controls for the four-SNP haplotypes tested in the other samples. The transmission distortion is unlikely to be due to chance, but its mechanism and specificity require further study. Our results illustrate the potential power of combining gene expression profiling and genomic analyses to identify susceptibility genes for genetically complex disorders.

L3 ANSWER 7 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2003:35175 BIOSIS
DN PREV200300035175
TI Current progress in ***schizophrenia*** research. Application of emerging "gene chip" technologies in search of genes.
AU Thaker, Gunvant K. [Reprint Author]
CS Maryland Psychiatric Research Center, Department of Psychiatry, University of Maryland School of Medicine, P.O. Box 21247, Baltimore, MD, USA
SO Journal of Nervous and Mental Disease, (November 2002) Vol. 190, No. 11, pp. 781. print.
ISSN: 0022-3018 (ISSN print).
DT Article
Editorial
LA English
ED Entered STN: 8 Jan 2003
Last Updated on STN: 8 Jan 2003

L3 ANSWER 8 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2002:627258 BIOSIS
DN PREV200200627258
TI Association and linkage analyses of ***RGS4*** polymorphisms in ***schizophrenia*** .
AU Chowdari, K. V. [Reprint author]; Mirnics, K. [Reprint author]; Semwal, P.; Wood, J. [Reprint author]; Lawrence, E.; Bhatia, T.; Deshpande, S. N.; Thelma, B. K.; Ferrell, R. E.; Middleton, F. A.; Devlin [Reprint author]; Levitt, P.; Lewis, D. A. [Reprint author]; Nimgaonkar, V. L. [Reprint author]
CS Department of Psychiatry, University of Pittsburgh, Pittsburgh, PA, USA
SO American Journal of Medical Genetics, (October 8, 2002) Vol. 114, No. 7, pp. 733-734. print.
Meeting Info.: Xth World Congress of Psychiatric Genetics. Brussels, Belgium. October 09-13, 2002. International Society of Psychiatric Genetics.
ISSN: 0148-7299.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 12 Dec 2002
Last Updated on STN: 12 Dec 2002

L3 ANSWER 9 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2002:624880 BIOSIS
DN PREV200200624880
TI Association and linkage analyses of ***rgs4*** polymorphisms in ***schizophrenia*** .
AU Kodavali, V. C. [Reprint author]; Karoly, M. [Reprint author]; Prachi, S.; Wood, J. [Reprint author]; Lawrence, E.; Bhatia, T.; Deshpande, S. N.; Thelma, B. K.; Ferrell, R. E.; Middleton, F. A.; Devlin, B. [Reprint author]; Levitt, P.; Lewis, D. A. [Reprint author]; Nimgaonkar, V. L. [Reprint author]
CS Department of Psychiatry, University of Pittsburgh, Pittsburgh, PA, USA
SO American Journal of Human Genetics, (October, 2002) Vol. 71, No. 4 Supplement, pp. 459. print.
Meeting Info.: 52nd Annual Meeting of the American Society of Human Genetics. Baltimore, MD, USA. October 15-19, 2002. American Society of Human Genetics.
CODEN: AJHGAG. ISSN: 0002-9297.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 12 Dec 2002
Last Updated on STN: 12 Dec 2002

L3 ANSWER 10 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2003:325835 BIOSIS
DN PREV200300325835
TI DISTRIBUTION AND DENSITY OF ***RGS4*** mRNA IN HUMAN POSTMORTEM BRAIN SECTIONS.
AU Lahti, R. A. [Reprint Author]; Erdely, H. A. [Reprint Author]; Lopez, M. B. [Reprint Author]; Roberts, R. C. [Reprint Author]; Tamminga, C. A. [Reprint Author]
CS Maryland Psychiat Res Ctr, Baltimore, MD, USA
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 703.2. http://sfn.scholarone.com. cd-rom.
Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience.

Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DT Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 16 Jul 2003
Last Updated on STN: 16 Jul 2003
AB RGS proteins are found throughout the body and are well distributed in the brain. There are about 20 RGS proteins, which are Regulators of G-protein Signaling. They enhance the rate of inactivation of the active form of the G proteins, by dramatically increasing the rate of hydrolysis of GTP to GDP. Of special interest was the finding that ***RGS4*** mRNA levels were decreased in the PFC of postmortem brain tissue obtained from ***schizophrenic*** subjects compare to normal controls (Mirnics, 2001). A more extensive distribution of ***RGS4*** mRNA has not been conducted in human postmortem tissue, and that is the aim of this study. The distribution and density of ***RGS4*** mRNA was determined in hemicoronal (Talairach sections +8 and -20) brain sections using in situ hybridization techniques. Highest levels were found in the more frontal sections (Talairach +8) and specifically in the inf. frontal ctx (73 nCi/gm), the sup. frontal ctx (71), the cingulate ctx (63); followed by the insular ctx and inf. temp ctx (51). The caudate (16), putamen (8.6) and nuc. acc. (3.5) had much lower levels. At Talarirch region -20, the cortical layers had the highest density (64.6 to 57.5), the parahippocampal gyrus had a density of 37.8, the CA-pyramidal region 16.3, and the thalamus was very low with a density of 2.3 nCi/gm. In the frontal cortex a dark band appears at one of the inner layers of the cortex. In conclusion, ***RGS4*** mRNA distribution in human postmortem tissue from normal controls was found to be very dense in most cortical layers, with much lower amounts in the basal ganglia and thalamus. This information will be used to guide a broad comparison between ***schizophrenic*** and normal control brain tissue.

L3 ANSWER 11 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:391331 BIOSIS
DN PREV200100391331
TI Analysis of complex brain disorders with gene expression microarrays: ***Schizophrenia*** as a disease of the synapse.
AU Mirnics, Karoly [Reprint author]; Middleton, Frank A.; Lewis, David A.; Levitt, Pat
CS Depts of Psychiatry, Neurobiology and PittArray, University of Pittsburgh School of Medicine, Pittsburgh, PA, 15261, USA
plevitt+@pitt.edu
SO Trends in Neurosciences, (August, 2001) Vol. 24, No. 8, pp. 479-486. print.
CODEN: TNSCDR. ISSN: 0166-2236.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 15 Aug 2001
Last Updated on STN: 22 Feb 2002
AB The level of cellular and molecular complexity of the nervous system creates unique problems for the neuroscientist in the design and implementation of functional genomic studies. Microarray technologies can be powerful, with limitations, when applied to the analysis of human brain disorders. Recently, using cDNA microarrays, altered gene expression patterns between subjects with ***schizophrenia*** and controls were shown. Functional data mining led to two novel discoveries: a consistent decrease in the group of transcripts encoding proteins that regulate presynaptic function; and the most changed gene, which has never been previously associated with ***schizophrenia*** , regulator of G-protein signaling 4. From these and other findings, a hypothesis has been formulated to suggest that ***schizophrenia*** is a disease of the synapse. In the context of a neurodevelopmental model, it is proposed that impaired mechanics of synaptic transmission in specific neural circuits during childhood and adolescence ultimately results in altered synapse formation or pruning, or both, which manifest in the clinical onset of the disease.

L3 ANSWER 12 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 4
AN 2002:221639 BIOSIS
DN PREV200200221639
TI Disease-specific changes in regulator of G-protein signaling 4 (***RGS4***) expression in ***schizophrenia*** .
AU Mirnics, K. [Reprint author]; Middleton, F. A.; Stanwood, G. D.; Lewis, D. A.; Levitt, P.
CS Dept of Psychiatry, University of Pittsburgh, E1602 BST, Pittsburgh, PA, 15261, USA
karoly+@pitt.edu
SO Molecular Psychiatry, (May, 2001) Vol. 6, No. 3, pp. 293-301. print.
ISSN: 1359-4184.
DT Article
LA English
ED Entered STN: 3 Apr 2002
Last Updated on STN: 3 Apr 2002
AB Complex defects in neuronal signaling may underlie the dysfunctions that characterize ***schizophrenia*** . Using cDNA microarrays, we discovered that the transcript encoding regulator of G-protein signaling 4 (***RGS4***) was the most consistently and significantly decreased in the prefrontal cortex of all ***schizophrenic*** subjects examined. The expression levels of ten other RGS family members represented on the microarrays were unchanged and hierarchical data analysis revealed that as a group, 274 genes associated with G-protein signaling were unchanged. Quantitative in situ hybridization verified the microarray ***RGS4*** data, and demonstrated highly correlated decreases in ***RGS4*** expression across three cortical areas of ten subjects with ***schizophrenia*** . ***RGS4*** expression was not altered in the prefrontal cortex of subjects with major depressive disorder or in monkeys treated chronically with haloperidol. Interestingly, targets for 70 genes mapped to the major ***schizophrenia*** susceptibility locus 1q21-22 were present on the microarrays, of which only ***RGS4*** gene expression was consistently altered. The combined data indicate that a decrease in ***RGS4*** expression may be a common and specific feature of ***schizophrenia*** , which could be due either to genetic factors or a disease-specific adaptation, both of which could affect neuronal signaling.

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Hale, Mary

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Sent: Tuesday, November 04, 2003 3:51 PM
To: Hale, Mary
Subject: RE: problem with search request for SN 09/939209

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Celine Qian
Art Unit 1636
CM1-11C-01
703-306-0283

-----Original Message-----
From: Hale, Mary
Sent: Tuesday, November 04, 2003 3:20 PM
To: Qian, Celine; Martinell, James
Subject: FW: problem with search request for SN 09/939209

Dear Examiner Qian -

You submitted a search request for SN 09/939209 Seq. 3 of that search has 20,300 residues, which is longer than our limit of 10,000 residues per sequence.

Your request has been been cancelled. Please meet with Jim Martinell. He will review your search request and will offer suggestions for modifying the search to optimize the processing time. Once you and he have met and made appropriate changes, please resubmit the search request so we can process your request immediately.

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308-4258, CM1-1E01

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  - ☐ 103 rejection
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  - ☐ Helped examiner better understand the state of the art in their technology.

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- ☐ Foreign Patent(s)
- ☐ Non-Patent Literature
  (journal articles, conference proceedings, new product announcements etc.)

- *Relevant prior art **not found:***
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  - ☐ Results were not useful in determining patentability or understanding the invention.

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